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ABSTRACT

In this literature review, the failure of a disease model of alcoholism based upon loss of control and craving phenomena to receive support is noted, as is the robustness of a model rooted in psychological learning theory. The viability of controlled drinking interventions based upon learning theory principles is demonstrated. Several predictor variables which emerged from studies using gamma alcoholics are identified: (1) low severity of dependence symptoms; (2) age 40 or less; (3) regular employment (favors a controlled drinking outcome); and (4) Alcoholics Anonymous involvement (favors an abstinent outcome). Possible learning theory explanations of these predictor variables are presented. Physical health/safety issues concerning controlled drinking research are also discussed. An apparently safe operational definition of alcohol consumption for future controlled drinking research of no more than 12.75 oz. ethanol equivalent/week, up to 2-5 oz. ethanol equivalent/drinking day is proposed. Other conclusions and suggestions for further research are offered. Seven pages of references conclude the report. (Author/NB)

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Doctoral Research Paper
for the Degree of Doctor of Psychology

ROSEMEAD SCHOOL OF PSYCHOLOGY

BIOLA UNIVERSITY

R. GLENN MOWBRAY

1986

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BY

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1986

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ABSTRACT

A REVIEW OF THE CONTROLLED DRINKING LITERATURE, WITH EMPHASIS UPON PATIENT VARIABLES DIFFERENTIALLY PREDICTING THE SUCCESS OF CONTROLLED DRINKING VERSUS ABSTINENCE OUTCOMES, AND PHYSICAL HEALTH/SAFETY ISSUES

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R. GLENN MOWBRAY

A review of the literature on controlled drinking was undertaken, with emphasis upon patient variables differentially predicting success for controlled drinking vs. abstinence outcomes, and physical health/safety issues. The failure of a disease model of alcoholism based upon loss of control and craving phenomena to receive support is noted as is the robustness of a model rooted in psychological learning theory. The viability of controlled drinking interventions based upon learning theory principles is demonstrated. Predictor variables which emerged from studies using gamma alcoholics are as follows: low severity of dependence symptoms; age 40 or less; regular employment (favor a controlled drinking outcome); AA involvement (favors an abstinent outcome). Possible learning theory explanations of these predictor variables are presented. Physical health/safety issues concerning controlled drinking research are also discussed. An apparently safe operational definition of alcohol consumption for future controlled drinking research of no more than 12.75 oz. ethanol

equivalent/week, up to 2-5 oz. ethanol equivalent/drinking day is proposed. Other conclusions and suggestions for further research are offered.

A Review of the Controlled Drinking Literature, With Emphasis Upon Patient Variables Differentially Predicting the Success of Controlled Drinking Versus Abstinence Outcomes, and Physical Health/Safety Issues

Introduction

Since 1962 (Davies) a controversy has existed in the field of alcoholism treatment. It began with the incidental report that a small percentage of alcoholics given treatment directed toward abstinence from alcohol were found to be drinking in moderation and in a stable fashion over time. This report sent shock waves throughout a field dominated by a disease model of alcoholism which holds that controlled drinking by alcoholics is an impossibility (Jellinek, 1960). In the intervening years since the initial furor, additional research has been performed in the area of controlled drinking. More than 100 studies documenting moderation outcomes from abstinence-oriented or moderation-oriented treatment programs have been conducted to date (Miller, 1983). While the controversy has continued on since 1962 in some circles, even as far as certain members of the scientific community leveling charges of fraud at two researchers and coverage of these charges by national media, the positive results contained in this growing body of literature make it increasingly difficult to question the validity and success of a controlled drinking intervention in alcoholism treatment (Miller, 1983; Heather & Robertson,

1981).

In light of this body of literature, it is time now to examine another phase of the research, that of patient variables which discriminate between the two treatments, and physical health/safety issues pertaining to controlled drinking. "Which patients do better in which treatment?" is the question being addressed here. Findings emerging from the literature relevant to this question will be discussed and conclusions will be presented.

History of Disease Conceptions of Alcoholism

This paper is about patient variables and physical health/safety issues salient to the treatment of alcoholism. Before proceeding to the empirical evidence which bears upon these matters, however, it is first necessary to undertake a review of the disease conception of alcoholism.

This review is necessary because the traditional treatment goal of total abstinence continues to be the major therapeutic response to the suffering caused by alcohol abuse. The claim that some alcoholics can return to non-problem or controlled drinking has been seen as controversial because it runs counter to this widely accepted and traditional treatment objective. For this reason a discussion involving controlled drinking must first consider the historical development of and theoretical justification for its pervasive alternative. This does not mean that these two solutions to the problem of alcoholism

are necessarily antagonistic or that they may not one day achieve a peaceful coexistence (cf. Jellinek, 1960, p. 77). But it does mean that the debate about controlled drinking vs. abstinence cannot be fully appreciated without first understanding why it is that abstinence has for so many years been regarded as essential to the alcoholic's recovery and why controlled drinking is considered impossible.

Early Disease Formulations

The origin of the disease theory of alcoholism is popularly thought to have coincided with the beginning of the alcoholism treatment movement, particularly at Yale University following the repeal of Prohibition in the United States. However, Jellinek (1960) and Levine (1978) make it clear that the modern understanding of alcoholism as a disease is rooted in late eighteenth and early nineteenth century thinking.

The core of the disease conception of alcoholism, which was worked out fully in the early years of the nineteenth century, was the concept of addiction. Two physicians, Benjamin Rush in America and Thomas Trotter in Britain, were mainly responsible for developing the new concept of addiction through the study of reports by many alcoholics that they experienced irresistible and overpowering desires for alcohol. Rush diagnosed alcoholism as a disease of the will and described the alcoholic's behavior in terms of a loss of control over drinking. Abstinence was prescribed by

Rush and others as the only cure for the disease. Rush is usually credited with being the founder of the Temperance movement in the United States and abstinence was enthusiastically adopted as the solution to alcoholism by the majority of temperance supporters.

Heather and Robertson (1981) point out an important difference between early and later disease conceptions of alcoholism. This has to do with the location of the source of addiction. In the temperance perspective, the origin of addiction was situated in the nature of the substance itself; anyone who drank alcohol was putting himself at risk of developing the disease. While there exist recent statements of the position that alcohol dependence is caused by nothing more than the regular and excessive ingestion of alcohol, more modern disease conceptualizations, including the Alcoholics Anonymous model of alcoholism, locate the source of addiction in the individual person. For a variety of hypothesized reasons, it is claimed that only some individuals are capable of becoming addicted to alcohol; moderate or heavy usage by those not so predisposed does not lead to addiction.

Despite this difference between the two versions of addiction, the present argument is that the core of modern disease conceptions of alcoholism, the concept of addiction itself and the loss of control over drinking which is alleged to follow from addiction, has been inherited from

early nineteenth century medical thought which gave birth to the Temperance Movement, and that the recent preference for abstinence as a cure for alcoholism has its roots in temperance ideology. Further, the organizational forms adopted by AA show it to be a modern counterpart of the temperance societies which flourished during the last century.

AA Model of Alcoholism

The AA model begins with the assertion of a qualitative difference between the alcoholic and all other types of drinkers. In the Big Book of AA (Alcoholics Anonymous, 1939), the "real" alcoholic is set apart from the moderate drinker who can live without it and from the hard drinker who, although he may be impaired physically and/or mentally through drinking, can stop or moderate it if he finds sufficiently strong motivation to do so. In contrast, the drinking of the real alcoholic is beyond such influence and cannot be stopped. The alcoholic is someone who, by definition, cannot do so. The basis for this difference lies in the supposed fact that the alcoholic is suffering from an allergy to alcohol. This idea comes from Silkworth (1937), one of the founders of AA, and was based upon observed similarities between the effects of alcohol and hay fever. Since the latter was an allergy the former must be also, it was reasoned. Haggard (1944) has dismissed this idea as having more to do with metaphor than medicine. More

recently, less emphasis has been placed on the allergy concept within AA and allusions are made instead to an as yet undiscovered physiological basis for the disease.

Alcoholics, then, according to this model are different from other people in that they cannot handle alcohol safely. Alcoholism is a disease, a progressive one, which can be arrested by total abstinence but which cannot be cured, it is believed. While medical science may one day discover the nature of the crucial physiological defect, this is not presented as a realistic possibility. Thus, those who attend AA call themselves recovering alcoholics even though they may have been abstinent for many years.

Another element which is apparent in the AA model is expressed in the idea that alcoholics have one symptom in common; they cannot start drinking without developing the phenomenon of craving (AA, 1939). Although accounts of the underlying cause may vary, craving or a compulsion to drink regardless of the consequences (Mann, 1968) appears as the subjective accompaniment of an objectively observable loss of control over drinking. Jellinek (1960) characterizes the AA view of loss of control as follows:

> Recovered alcoholics in AA speak of 'loss of control' to denote that stage in the development of their drinking history when the ingestion of one alcoholic drink sets up a chain reaction so that they are unable to adhere to their intention to 'have one or two drinks only' but continue to ingest more and more--often with quite some difficulty and disgust--contrary to their volition. (p. 41)

AA thinking may have changed in certain respects over

the years, but when it comes to the loss of control hypothesis there has been no change whatsoever. Thus is the necessity for lifelong abstinence on the part of AA ideology among the general public. Abstinence is made all the more imperative by the fact that alcoholism is said to be a progressive disease; if drinking is continued, further deterioration is inevitable.

It would be difficult to overestimate the influence of AA on medical theory and practice in the field of alcoholism. The fellowship has always been interested in gaining friends among the medical profession, as several AA pamphlets attest (Alcoholics Anonymous, 1955; 1965). This effort has been remarkably successful. Many hospital alcoholism treatment programs consist of little more than a formalized version of AA principles or make attendance at AA meetings a part of treatment regimen. Jones and Heldrich (1972) found physicians who agreed that alcoholism was a disease generally believed that referral to AA was an important part of treatment.

Jellinek's Disease Conception

Jellinek (1952) was a seminal thinker in the field of alcoholism whose ideas exert tremendous influence currently. He began by applying the concept of alcoholism to excessive drinkers characterized by physical or psychological problems. Among those thus identified as alcoholics, there are two distinct types, according to Jellinek. These were

"alcohol addicts" to whom the disease concept of alcoholism applies and "habitual symptomatic excessive drinkers" to whom it does not. The criterion separating these types is "loss of control" (p. 679) which develops only among alcohol addicts after many years of excessive drinking. Once it has developed, it can manifest itself after a few hours or weeks of drinking, and lasts until the drinker is too intoxicated or sick to continue. Jellinek believed the habitual symptomatic excessive drinker is also a sick person, but that the basic problem is the psychological or social difficulties from which drinking gives temporary relief. This basic dichotomy of alcohol addicts and habitual symptomatic excessive drinkers also reflects the AA distinction between real alcoholics and hard drinkers.

The alcohol addict but not the less serious types of problem drinkers pass through a series of phases. In the Prealcoholic Symptomatic Phase, drinking progresses from occasional to constant relief-drinking. The sudden onset of alcoholic blackouts marks the beginning of the Prodromal Phase which is marked by surreptitious drinking, preoccupation with alcohol, and the occurrence of guilt feelings. Jellinek stated that even at this phase the only possible solution for the alcoholic is abstinence.

The Crucial Phase of addiction is defined by the onset of loss of control. Jellinek, like AA saw loss of control in terms of an inability to stop once drinking had

commenced. The Crucial Phase is succeeded by the Chronic Phase in which alcohol tolerance is reduced and the person becomes entirely incapacitated.

It is interesting to note that Jellinek's conception of alcoholism, incorporating as it does the use of alcohol for the relief of psychological stress, is compatible with the tension reduction/reinforcement model (Conger, 1956) which was developed shortly after Jellinek's (1952) ideas were published. Heather and Robertson (1981) state that Jellinek's formulation could accurately be described as a learning theory of alcoholism were it not for the crucial component of loss of control. It follows that the justification for his disease conception of alcoholism turns on the empirical validity of this concept. Loss of control ceases to be merely a symptom of alcoholism and becomes a construct upon which the entire disease conception depends.

In his 1960 book, in order to do justice to international as well as American data he had collected on alcoholism, Jellinek broadened his operational definition of alcoholism to "any use of alcoholic beverages that causes any damage to the individual or society or both" (p. 35). This book is best known for its Greek letter classification concerning the five proposed species of alcoholism: viz., alpha, beta, gamma, delta, and epsilon.Jellinek proposed that only the gamma and delta species are diseases, the reason being that only they entail physiological changes

analogous to those in drug addiction (adaptation of cell metabolism, acquired increased tissue tolerance and withdrawal symptoms). The gamma type alcoholic cannot stop drinking once it is begun due to craving and loss of control. The delta type cannot refrain from initially starting to drink. The physiological changes are supposedly responsible for bringing about loss of control (or inability to abstain), craving, and inability to refrain. In 1960, Jellinek thus firmly ties in his disease conception of alcoholism with drug addiction in general and endorses a neurophysiological substrate for addiction and loss of control.

Jellinek is also more specific about the postulated mechanism of loss of control. It is said to occur in gamma alcoholics as a result of minor withdrawal symptoms in the presence of diminishing amounts of alcohol in the bloodstream. Continued drinking is necessary in order to abolish these unpleasant dependence or withdrawal symptoms, which include tremors of the fingers and lips, slight twitchings, motor restlessness, and sometimes delusions. The drinker achieves some degree of temporary relief, but only for a short period before the withdrawal symptoms recur and further drinking becomes necessary. The demand for alcohol is of a twofold nature. First, there is the physical demand brought about by the need to allay distressing withdrawal symptoms (loss of control), and

secondly, there is the accompanying obsessive belief, known as craving, based on the alcoholic's experience before loss of control became established to the effect that continued drinking will eventually provide a reduction of tension.

As in Jellinek's original 1952 formulation, it is noted that the sole justification for his disease conception of alcoholism is the notion of loss of control. In the case of gamma alcoholism the physiological processes signaling the onset of addiction are identical with those used to explain loss of control and the two concepts are therefore synonymous. Therefore, loss of control in one sense or another is the premier notion in Jellinek's disease conception and "without it his model would become a learning theory of alcoholism incorporating individual differences in the tension-reducing effects of alcohol and supplemented by a sociocultural perspective" (Heather & Robertson, 1981, p. 13). Following his 1960 book, his conception of alcoholism has become dominant in medical and psychiatric circles. His view of alcoholism as a disease characterized by a form of addiction based on physiological changes which are caused in turn by prolonged excessive drinking, has been widely adopted.

This concludes the review of the history of the disease model of alcoholism based upon loss of control and craving formulations and the theoretical justification of abstinence as the only treatment possibility within this disease model.

Following is a review of research looking at the notions of loss of control and craving. Weaknesses in the disease model as a result of the failure of these notions to hold up under scientific experimentation will be discussed and learning theory alternatives to the disease model will be presented.

Loss of Control and Craving: Research

As has just been shown, the concept of loss of control and the associated notion of craving are the "sine qua non" of a disease conception of alcoholism. Loss of control is the pivotal concept in Jellinek's progression from relief drinking to alcoholism. The concept is also essential to AA theorizing. Keller states that it is the "pathognomonic sign of alcoholism" (1972, p. 153). Jellinek considered the concept a working hypothesis marking entrance to the third phase of alcoholism in which the mere ingestion of even small amounts of alcohol (one or two drinks) would once again bring on a state of physical dependence (Pattison, Sobell, & Sobell, 1977). Following is a group of experimental studies relevant to the disease notions of loss of control and craving. In this literature, a number of descriptions are used to convey measurement of the amount of alcohol consumed. For this and the following sections these various descriptions have either been given with or else converted into equivalent ounces of absolute alcohol (ethanol), with .5 oz. ethanol equalling approximately 1 oz.

100 proof whiskey or one 12 oz. can of beer.

Mello and Mendelson (1965) conducted an experiment in which two adult male alcoholic subjects with a 20 year history of alcoholism and a history of dependence symptoms consistent with a diagnosis of gamma alcoholism were given an opportunity to work with an operant conditioning device for two weeks. The subjects had free access to the device and could choose to work for either 10 ml. whiskey (.17 oz. ethanol equivalent) or for money by turning a switch indicating their choice. When the whiskey-containing glass was removed the entire device shut down for 10 minutes. The response required was to press a lighted key according to a complex schedule of reinforcement. Both subjects preferred to work for alcohol rather than the money. They also drank steadily throughout the course of the experiment. By working periods of between one and three hours/day they were able to maintain a blood alcohol concentration (BAC) of between .15 and .25%, which corresponds to several drinks per hour. Subjects did not work to amass huge quantities of alcohol nor did they drink to gross intoxication during a given period of drinking. Instead, they chose to work steadily for small amounts and drink these slowly over time. These results run counter to predictions based upon loss of control and craving formulations.

Similar findings were obtained in a follow-up study (Mendelson and Mello, 1966) in which the key pressing task

was replaced with the more interesting one of controlling a model car on a track. Points were earned by keeping the car on the track. These could be exchanged later for either alcohol or money. Subjects in this study also drank in a manner which maintained stable BAC's despite essentially unlimited access to alcohol. They did not become grossly intoxicated but displayed only mild intoxication without marked speech or motor impairment.

In a further study (Mello, McNamee, & Mendelson, 1968), the experimenters were interested in assessing what effect cost of alcohol would have on drinking behavior. Cost was operationalized as the amount of effort expended to obtain a drink. Two groups were used in a task in which a button had to be pressed within .5 sec. of the appearance of a stimulus light. The first group was required to make 16 consecutive successful presses and the second group 32 presses in order to receive the alcohol. It was found that BAC's in the first group were roughly twice as high as those in the second group, showing that the amount of alcohol consumed is a function of the amount of work required to obtain it. Subjects in both groups worked for moderate amounts of alcohol and consumed it in a modulated fashion over time. In addition, several of the subjects, realizing that the experiment would be over soon, on their own began cutting back on their intake in order to decrease the likelihood of experiencing withdrawal symptoms at the conclusion of the

experiment. All of these results point to the existence of various kinds of control over drinking behavior in these alcoholic men.

Mello and Mendelson (1972) wanted to assess drinking patterns of male alcoholics over a longer period than previously studied. Most of the subjects consumed about 1 qt. whiskey/day and reported moderate to severe withdrawal symptoms upon cessation of drinking. In one part of the experiment, 2 groups of 4 subjects were studied for periods of 30 and 62 consecutive days, respectively, in a task of pressing a button 1,000 times. Upon completing this task, a token was received which could be traded for 1 oz. (.5 oz. ethanol equivalent) of whiskey or a cigarette. All eight subjects showed a clear demarcation between times of work and times of drinking. The subjects would work without drinking for one or two days, then drink without working for the same amount of time. This cycle repeated itself a number of times over the course of the experiment. During abstinent times the men frequently exhibited withdrawal symptoms, ranging in severity from mild to moderate. The authors note that in spite of these withdrawal symptoms the men did not choose to abolish them during a working phase by resuming drinking, even though they were free to do so. Rather, they preferred to go back to collecting tokens. This finding is contrary to a loss of control formulation which would predict that the subjects would not be able to

stop their drinking.

The studies reviewed so far show that male alcoholics who are pharmacologically dependent, who were in situations where they could freely determine the amount and timing of their drinking, did not lose control and drink to states of unconsciousness or collapse. Any existing cravings or compulsions to drink did not lead inevitably to severe intoxication. Evidence of various forms of control were commonly observed. High but stable BAC's over time were the rule. Some subjects chose to taper off their drinking in anticipation of the ending of an experiment in order to reduce withdrawal symptoms. Subjects chose to work to accumulate alcohol rather than drink to abolish partial withdrawal symptoms. All of these findings are inconsistent with a definition of loss of control as the inability to stop drinking once it has started, and with the related notion of craving as an irresistible urge to drink more and more alcohol during a drinking session.

Further, the finding in a free choice situation that the amount of alcohol consumed was a function of the amount of work required to obtain it shows that alcoholic drinking is an operant behavior (Heather & Robertson, 1981). That is, drinking behavior can be shaped and maintained by environmental contingencies. Given the evidence cited above in support of contingency control of drinking and the failure of a disease model predicated upon a loss of control

or craving to be supported, a learning theory approach to the problem of alcoholism may be more appropriate.

Following is a group of studies in which other types of environmental contingencies and their effect on drinking behavior were examined. Cohen, Liebson, Faillace, and Speers (1971) also focused upon cost factors in alcoholic drinking. Unlike Mello et al. (1968) and Mello and Mendelson (1972) though, the target behavior was abstinence and the experimenters were assessing what reinforcement contingencies were required to obtain abstinence. The four male, chronic alcoholic subjects were allowed to purchase relatively large quantities of alcohol (12 oz. ethanol equivalent) every third day of the experiment. On following days each was offered money to abstain for the entire day. If the subject did not abstain the incentive was increased for the next occasion, but if he abstained, it was decreased. It was determined that abstinence could be bought for $7-$20. The first experimental manipulation was concerned with delay in reinforcement. Subjects were offered the same amount for which they had previously abstained but this time payment was delayed for from three to twenty-one days. Again, payment was increased if the subject drank but in this condition the delay in reinforcement was increased if he abstained. Results showed that delay in reinforcement disrupted abstinence. But an increase in payment was effective in reinstating it. The

same results were obtained in the second experimental manipulation when a priming dose of alcohol was administered on the morning of the day on which payment for abstinence was offered. A priming dose of alcohol tended to disrupt abstinence for subjects but increasing the monetary reinforcement reestablished it.

This experiment offers a possible explanation as to how a belief in loss of control could have arisen (Heather & Robertson, 1981). Consistent with this notion, an alcoholic who has consumed alcohol could reject abstinence even though it has reinforcing consequences and even though he might have accepted it when initially sober. The point of this experiment, however, is that drinking could be brought back under control once again by providing a sufficiently large positive reinforcement, showing loss of control to be a transient phenomenon. Delay in reinforcement is also highly relevant to the issue of loss of control, since in the real world it can be assumed that positive reinforcers for the alcoholic remaining abstinent are more remote than such reinforcement for drinking. In spite of this, the study shows alcoholics will tolerate relatively long delays in reinforcement given that the incentive for abstinence is sufficiently large.

Cost was also the object of study for Bigelow and Liebson (1972). Two male, skid row, chronic alcoholic inpatients were used as subjects. In the first part of the

experiment, receiving a drink (.5 oz. ethanol equivalent) was contingent upon a fixed ratio of lever-pulling responses. Results showed that when 3,000 responses/drink were required, subjects moderated their drinking as compared to lower ratios, but did not choose to abstain. At a ratio of 5,000, however, subjects occasionally chose to abstain rather than exert the required amount of effort. Essentially the same results were obtained in the second part of the study in which tokens were used to purchase drinks. As the price/drink increased, the consumption of alcohol decreased. Alcoholic drinking was not shown to be uncontrollable but could be brought under control by environmental influences. The authors conclude that cost in the form of physical effort, and economic cost in a token condition were both efficacious in modifying alcoholics' drinking behavior.

A different class of reinforcer was studied by Cohen, Liebson, and Faillace (1972). Being permitted in an enriched vs. impoverished hospital environment was made contingent upon moderated drinking for five male gamma alcoholics. The experiment, using an A-B-A-B-A design, demonstrated clearly that alcoholic drinking behavior can be brought under control by a variable with relevance to the natural environment.

Another reinforcer was investigated by Bigelow, Liebson, and Griffiths (1974). Ten male, chronic alcoholic

patients were given daily free access to alcohol (6-12 oz. ethanol equivalent) during a baseline period. During the following experimental condition, however, the immediate consequence of taking a drink was "time-out" from social reinforcement. In time-out, subjects were isolated for 10-minute intervals. Additionally, a reversal condition was included following the time-out contingency. Results showed that drinking was suppressed to roughly half its baseline level for seven of the ten subjects. During the baseline, subjects consumed an average of 94.6% of the alcohol available but in the contingency condition this fell to 52.1% ($p = .0001$). In the reversal, drinking once again returned to over 90% of the quantity available. The authors note that the results of this study may seem surprising in light of the observation sometimes made in the natural environment of alcoholics failing to modify their behavior in spite of the loss of multiple reinforcers (e.g., job, marriage, friends). They believe, however, the difference between modifying and failing to modify drinking behavior lies in the immediacy and reliability of the loss of reinforcement.

The results of the previous studies are important because they show the administration and withdrawal of an enriched environment and social reinforcement are effective in modifying drinking behavior in chronic alcoholics. This is contrary to the traditional disease view based upon a

loss of control formulation that drinking by alcoholics is a relatively automatic activity unresponsive to environmental and social factors.

The experiments discussed so far in this section deal with the modifiability of alcoholic drinking behavior via operant conditioning procedures. They show that little support exists for a disease model based upon loss of control or craving. The following experiments deal with attempts to separate out physiological from psychological components of loss of control and craving. Merry (1966) reported on a study in which nine inpatients diagnosed as gamma alcoholics were given a "vitamin drink," consisting of 1 oz. vodka (.5 oz. ethanol equivalent) mixed with orange juice. Previous testing had shown that this dosage of alcohol was undetectable. The control condition used water instead of vodka mixed with the juice. Subjects acted as their own controls and were given either mixture at breakfast in 2-day sequences over 18 days. During the late morning of each day subjects were asked to rate on a 5-point scale the amount of craving experienced. After 63 administrations of the vodka mixture, craving reactions occurred only 15 times, and after 72 of the nonalcoholic administrations they occurred 20 times. The results showed no difference in average craving scores between alcoholic and nonalcoholic mixtures. Merry interpreted these results as failing to support the hypothesis that small quantities

of alcohol trigger off a biochemical abnormality leading to craving in gamma alcoholics and concluded that psychological and environmental factors were more important.

Paredes, Ludwig, Hassenfeld, and Cornelison (1969) gave 66 chronic male and female alcoholics a 3 or 4 oz. dose of vodka (1.5-2.0 oz. ethanol equivalent) before interviewing them in a study involving self-image. An incidental finding of this study is that only three of the subjects reported cravings for alcohol after drinking the initial dose and none of the seventeen subjects followed-up on an outpatient basis the next week had continued to drink with loss of control. The authors concluded that social setting and cognitive factors (e.g., the meaning of drinking in this setting) might be salient in the induction of craving.

Engle and Williams (1972) studied four groups of ten alcoholic inpatients (32 men; 8 women). Groups 1 and 2 were given a "vitamin mixture." Group 2's mixture contained 1 oz. of vodka (.5 oz ethanol equivalent) without subjects being aware of this, however. Groups 3 and 4 were also given the mixture but were told it contained alcohol. Only for group 3 was this true, though. The nurse administering the mixtures was blind as to their content. Craving scores were obtained as in Merry (1966) above. The only significant difference between group means was between groups 2 and 3 ($p = .02$), with group 2 obtaining the lower mean. Both groups had actually received alcohol but the

group not so informed obtained the lowest mean craving score. The group correctly informed obtained the highest mean score. Engle and Williams interpret these results as indicating that an alcoholic's craving for alcohol increases when he has been informed he has drunk alcohol, whether in fact he has or not. These results therefore support a cognitive psychological interpretation of craving and provide no support for a physiological relationship between alcohol and craving.

Marlatt, Demming, and Reid (1973) employed a design similar to Engle and Williams (1972). However, they used a population of social drinkers as controls in addition to the experimental group of chronic alcoholics. Also, dependent measures were directed more to loss of control than to craving. Results were that alcoholics drank more than social drinkers and the main determinant of amount consumed was instructional set; for both groups, those told they were drinking alcohol drank more. The authors conclude that their findings provide support for the role of cognitive factors in the determination of loss of control drinking.

The implication of these experiments is that drinking behavior typically described as loss of control or craving seems to be mediated by cognitive processes and not by a biochemical reaction to ethanol. Maisto, Lauerman, and Adesso (1977) point out, though, that since it is impossible to prove the null hypothesis, it would be an error to say

that a physiological basis for loss of control had been disproved. Rather, a more appropriate conclusion would be that these experiments provide no evidence for the existence of a physiological basis for loss of control.

Because of the evidence accumulating from the experiments mentioned above, defenders of a disease notion based upon loss of control and craving have been forced to make certain revisions in their theorizing. Keller (1972) took issue with Jellinek's definition of loss of control as the inability to stop once drinking has started. Keller reconceptualized loss of control as both an inability to stop once drinking has started and an inability to refrain from beginning to drink. This amounts to a merging of Jellinek's original distinction between the gamma and delta alcoholics. What is also interesting is that Keller makes an appeal to learning theory. He proposed a redefined concept of loss of control as a conditioned drinking response elicited by environmental stimuli. Stimulus generalization is said to account for the spread of addictive conditioning, leading to binges. Heather and Robertson (1981) see a problem with Keller's use of learning theory to shore up his revised theory of loss of control. They point out that he tries to defend the disease conception of alcoholism by pointing to a sense in which the alcoholic's drinking is outside his choice because it is conditioned. The implication then is that the drinking of

nonalcoholics is not conditioned. They conclude that Keller misuses learning theory which would insist that all drinking, and not just the drinking of alcoholics, is learned behavior and subject to conditioning processes.

The use of classical conditioning as an explanatory process in the resumption of drinking suggested by Keller was elaborated by Ludwig, Wikler and Stark (1974) in their notion of the "subclinical conditioned withdrawal syndrome." According to this theory, with the development of physical dependence on alcohol, withdrawal phenomena become classically conditioned to certain stimuli, such as the physical environment in which alcohol is consumed, drinking associates, and induced emotional states, through a process of repeated temporal contiguity between these stimuli and the unconditional withdrawal symptoms. Also, alcohol-seeking behavior is reinforced by the reduction of withdrawal symptoms following alcohol consumption in a process of operant conditioning. Following a period of refraining from drinking, exposure to the same or similar stimuli then evoke conditioned withdrawal feelings, leading to alcohol-seeking behavior and relapse. Heather and Robertson (1981) extensively criticize the experiment, citing statistical and methodological flaws and disagree with the experimenters over how much support was generated for their hypotheses. In spite of this, they conclude that the notion of a subclinical withdrawal syndrome, if

restricted to the conditioning of withdrawal symptoms, does seem to have some merit.

Conclusions From Loss of Control and Craving Research

Two conclusions can be made from this review of research on loss of control. First of all, a disease theory of alcoholism based upon a loss of control formulation with its attendant notion of craving failed to receive support. Rather, support exists for a psychological interpretation of loss of control and craving phenomena, based particularly upon operant and classical conditioning models and cognitive psychological explanations. Secondly, keeping Maisto et al. (1977) in mind, it cannot be said that a biochemical or other physiological basis for loss of control phenomena has been ruled out. However, at this time a more congruent interpretation of the data would favor a psychologically-based understanding of the phenomena.

Controlled Drinking Research

Heather and Robertson (1981) review 16 studies involving controlled drinking interventions with a hospitalized population of alcoholics. Using a stringent set of methodological criteria, nine of these studies were considered inviable by them. The criteria used were: follow-up of at least six months; drop-out rates for comparison groups within 10% of each other; blind assessment; random assignment; control groups comparable to treatment groups on important variables; and use of

systematically derived corroborative evidence from collaterals, blood tests or official records. In addition to the criteria used by Heather and Robertson, the author has used the additional one of including only those studies using clearly defined gamma alcoholics or alcoholics who would likely receive this diagnosis (chronic hospitalized alcoholics). This is necessary in order to preserve theoretical continuity between the loss of control research previously presented, the controlled drinking research at hand, and the predictor variable studies presented later. It is the gamma alcoholic for whom loss of control applies and for whom traditionally the only treatment is abstinence. Therefore, in considering the viability of a controlled drinking intervention, only gamma alcoholics should be considered. That alpha, beta, and delta types might also benefit from a controlled drinking intervention is clinically important but theoretically tangential to this discussion. Six studies remain and these will be discussed.

Caddy and Lovibond (1976) used 60 hospital patients (49 males; 11 females), most of whom had been hospitalized several times for alcoholism. Each of the subjects was randomly assigned to one of three groups. The first group received avoidance conditioning plus self-regulation treatment which consisted of BAC feedback training, training in self-regulation, psychotherapy, and general education in the belief that drinking should and could be moderated. The

second and third groups received the same treatment as the first group except that for the second group no shock was used, and for the third group self-regulatory procedures were not emphasized. Outcome was measured through self- and other-reports of daily alcohol intake (these results were not published) and BAC. Assistants who were blind to treatment group membership assigned subjects to one of three outcome categories ("completely successful in controlling drinking," "moderately improved," or "slight/no improvement"). Follow-up information was available on all subjects at 6 months and on 62% at 12 months. Tests of statistical significance were performed only on the 6-month follow-up data, however. Results show that the majority of subjects for all treatment groups were in either the "completely successful" or "moderately improved" outcome categories and that the first group, avoidance conditioning plus self-regulation treatment, was superior to either of the other two treatment groups ($p = .05$). While this study did not include a no-treatment or abstinence control group to assess for the absolute effects of a controlled drinking intervention, a differential treatment effect was clearly demonstrated, showing that the combined treatments were more effective than either one alone.

At Patton State Hospital, Baker, Udin, and Vogler (1975) used 40 male, alcoholic inpatients as volunteers. All subjects had experienced alcohol withdrawal symptoms.

The subjects had an average of 4.4 previous hospitalizations for alcoholism and an average 10.9 year history of problems due to alcohol. Subjects were randomly allotted to one of four treatment groups. A group of controls received standard hospital treatment, which included group therapy and AA meetings. The three remaining groups all received three sessions of behavioral counseling, consisting of training in alternative responses to cues known to elicit heavy drinking. Also, subjects in these three groups were given information about appropriate drinking styles and regulated drinking practice. The second of these remaining groups in addition received videotape self-confrontation of their intoxicated behavior. The third of these remaining groups viewed "modeling" films in which the negative consequences of excessive drinking and the positive consequences of moderate drinking were portrayed. Outcome was measured six months after treatment by estimating the number of days abstinent or drinking in a controlled way. This information was obtained principally by self-report. Other-reports (friends, relatives, or a minister) and a state print-out service listing later institutional admissions were used to a lesser extent. Excessive drinking was defined as the consumption of 14 oz. or more of 86 proof liquor or its equivalent (7 oz. ethanol equivalent) over any consecutive 2-day period. No significant differences between treatment groups emerged. Success was defined as

the experience of fewer than 20% days of excessive drinking and by this criterion 27.5%, or 11 out of 40 subjects, were successful.

Vogler, Compton, and Weissbach (1975) studied 42 Patton State Hospital inpatients (37 males; 5 females) with a mean of 5.96 alcohol-related arrests, a mean 11.08 year history of problem drinking, and a mean pretreatment consumption rate of 5.15 oz. ethanol equivalent/day. Subjects were randomly assigned to two groups, the first receiving alcohol education, regulated drinking practice, BAC discrimination training, avoidance conditioning, and "behavior counseling" (stimulus control, problem-solving skills training, assertiveness training, relaxation training, social skills training, and videotape feedback of their behavior after drinking). The second group received alcohol education and behavior counseling only. Booster sessions were given each month for 12 months. Assessors blind to the experimental manipulation collected numerous outcome data, including alcohol intake, number of days lost at work, number of days drunk, number of days drinking, and amount spent on alcohol purchases. Follow-up information was from self- and other-reports (family and friends), especially when there were discrepancies in the self-reports. No significant differences emerged between groups. However, confounding exists because the second group had a lower pretreatment alcohol intake. This suggests that the first group may

actually have improved more than the second but this cannot be stated definitely. Of the total 42 subjects, 12 were drinking in a controlled manner (average intake less than 1.7 oz. ethanol equivalent/day and no more than one episode of uncontrolled drinking, defined as a BAC exceeding 80mg/100ml), 16 had relapsed, and 14 were abstinent.

Sobell and Sobell (1973; 1976) carried out the most extensive study to date on controlled drinking. Subjects were 70 male, gamma alcoholics at Patton State Hospital who volunteered to participate in a research program. The study consisted actually of two separate studies, the first of which is of interest to this discussion. In this study, subjects were assigned either to an experimental group which received 17 sessions of "individualized behavior therapy" with the goal of controlled drinking, or a control group which received conventional abstinence-oriented treatment. Individualized behavior therapy consisted of videotape replay of drunken behavior, electrical avoidance conditioning, stimulus control training, problem-solving skills training, and regulated drinking practice. The control group received group therapy, chemotherapy, AA, and physiotherapy.

Outcome measures included self-reports of daily consumption, ratings on a 3-point rating scale by a friend, relative, or employer of the subject's interpersonal relationships and life adjustment, occupational and

vocational status ratings, outpatient therapy sessions obtained, and number of days spent in a hospital or jail. A controlled drinking day was defined as one in which 3.0 oz. ethanol equivalent/dat or less were consumed, or any isolated 1 or 2 day sequence when between 3.5 and 4.5 oz. ethanol equivalent/day were consumed.

Follow-ups occurred at the end of the first and second years. A 3 year follow-up was also conducted by a separate group unaware of the group status of subjects (Caddy, Addington, & Perkins, 1978). At two years, information on 69 of the original 70 subjects was obtained and at three years on 53 subjects.

Significant differences ($p < .01$) were obtained on the following dimensions. On the measure "percentage days drunk" differences between treatment and control groups in the second and third years of follow-up were 12.3 vs. 49.2% and 5.1 vs. 25.3%. On "percentage days abstinent" a difference was obtained in year two (62.6 vs. 36.5%). Differences in "percentage of days spent drinking in a controlled manner" for the first and second years were 25.2 vs. 9.6% and 22.6 vs. 5.8%. On the dimension of "percentage days functioning well" (either abstinent or controlled drinking) differences resulted in the first and second years (70.5 vs. 35.2%; 74.5 vs. 44.9%). The measure of "vocational status" (percentage improved on this measure) yielded differences in year two (70.0 vs. 28.8%).

Differences were obtained on "occupational status" (percentage employed) in years one and two (76.9 vs. 46.7%; 75.0 vs. 53.5%). In terms of "general adjustment" (percentage improved) differences resulted for both years one and two (85.0 vs. 34.2%; 87.5 vs. 44.7%). The measure was not taken for year three. Finally, on the "general index of outcome" differences in group mean scores were obtained for years two and three (.86 vs. .48; .84 vs. .66). This measure was not taken for the first year. No significant differences were obtained on the measure of "percentage days incarcerated in a hospital or jail." In all instances which failed to reach significance, the trend was in the direction of the treatment group being superior to the control group. Also, in almost all cases there was improvement over time for both treatment and control groups, with the treatment group generally posting more marked improvements.

It is noted that Sobell and Sobell (1973; 1976) and Caddy et al. (1978) obtained different results than Baker et al. (1975) in similar studies. Possible reasons for this are that the former studies utilized a greater number of behaviorally-oriented training sessions (17 vs. 3), a longer follow-up period (1-3 years vs. 6 months), and a more sophisticated reporting system on subject drinking behavior which obtained information from collaterals for all subjects, rather than just a few.

Media Involvement

The original study (1973) and follow-up (1976) by Sobell and Sobell have been the focus of attack by both certain members of the scientific community and national media. On June 28, 1982, Irving Maltzman, referring to these studies, was quoted in The New York Times as saying, "Beyond any reasonable doubt, it's fraud." (Boffey, 1982, p. A12). Maltzman was co-author of an article published soon afterwood in the July 9, 1982 issue of Science (Pendery, Maltzman, & West, 1982) based upon his group's independent 10 year follow-up of the original subjects in the Sobells' 1973 study. It was claimed in this article that in the Sobells' original study most subjects trained to do controlled drinking failed from the outset to drink safely and were re-hospitalized within one year of discharge from the hospital. Also it was charged that the experimental and control groups were not randomized. As a result of these published reports, some individuals suspected that the data of the original 1973 study must have been falsified or seriously misrepresented.

In response to these allegations of professional misconduct, in June of 1982 the Addiction Research Foundation in Toronto appointed a panel of independent investigators to study the issue. Headed by Bernard Dickens, the results were released in a final report on November 5, 1982 (Dickens, Doob, Warwick, & Winegard, 1982).

The conclusion is stated on p. 109:

> The Committee has reviewed all of the allegations made against the Sobells by Pendery et al. in their published Science Article, and in various statements quoted in the public media. In response to these allegations, the Committee examined both the published papers authored by the Sobells as well as a great quantity of data which formed the basis of these published reports. After isolating each of the separate allegations, the Committee examined all of the available evidence. The Committee finds there to be no reasonable cause to doubt the scientific or personal integrity of either Dr. Mark Sobell or Dr. Linda Sobell.

A major flaw in the Pendery et al. (1982) study is that 10 year follow-up results are discussed only for the controlled drinking group. Data for the abstinence-oriented control group are omitted. Since the only meaningful comments from a 10 year follow-up would be those stemming from a statistical comparison of outcome data for both groups, the results are uninterpretable.

On March 6, 1983 the CBS television show 60 Minutes featured a report critical of the Sobell and Sobell (1973) research in which the narrator, Harry Reasoner, was shown at the gravesite of one of the former controlled drinking subjects. The message was clear: controlled drinking kills (Marlatt, 1983). Although Pendery et al. (1982) reported that 4 of the original 20 controlled drinking subjects eventually died alcohol-related deaths, neither these authors nor Harry Reasoner mentioned that 6 of 20 abstinence-oriented control subjects also died from the same causes over the same time period.

It can be seen from the above discussion that studies in controlled drinking have been the focus of much controversy in recent years. However, it must be concluded that no evidence exists for charges of impropriety on the part of the Sobells or any other researchers in the area of controlled drinking.

Conclusions from Controlled Drinking Research

Several conclusions can be drawn from the studies reviewed in this section. First of all, a controlled drinking intervention utilizing behavioral techniques can be quite successful compared to a non-behavioral, abstinence-oriented treatment modality. Secondly, further research is needed to differentiate between which types of behavioral interventions, if any, are relatively more successful than others. Some evidence exists that multiple rather than single behavioral interventions are more successful. Thirdly, further research is needed in the application of behavioral techniques to abstinence-oriented treatment and in comparing controlled drinking and abstinence treatments based upon behavioral techniques. Fourthly, research to date has either not included female subjects or else included only small numbers of them. As a result, it is unclear whether the experimental results have general applicability to both sexes. Future research should include greater numbers of women to test this possibility.

Selection of Candidates for Controlled Drinking versus Abstinence Outcomes

Given that a controlled drinking intervention is a viable alternative to abstinence, a relevant question to ask next would be, "For whom controlled drinking?". A number of factors must be considered when assessing whether a particular person should be considered for a controlled drinking vs. abstinence program (Miller & Caddy, 1977). First of all, the person's wish to either learn to drink in a controlled manner or not drink at all is a very important consideration. Heather and Robertson (1981) summarize the results of four studies in which clinic alcoholics could choose either controlled drinking or abstinence treatments. Between 15 and 37% of the inpatients chose to learn controlled drinking. The existence of liver disease or other serious health problems would contraindicate controlled drinking. Bizarre or aggressive behavior following even moderate alcohol use would also rule out controlled drinking. The prior failure of therapy oriented toward either controlled drinking or abstinence would be a reason to consider the alternative treatment. Also important would be a consideration of the person's lifestyle, peer group affiliation, and vocation (e.g., a businessman who is expected to drink with clients).

Predictor Variable Studies

In addition to these considerations, research has been performed which attempts to discover variables which predict success or failure in controlled drinking vs. abstinence outcomes. This research is important because it represents a further attempt to identify those persons for whom either controlled drinking or abstinence may be appropriate. Twelve studies bear on this issue of predictor variables and will be reviewed. Heather and Robertson (1981) conducted a similar review but the author eliminated a number of the studies they included on methodological and theoretical grounds, which will be discussed below. It should be remembered, as these studies are reviewed, that half involved a treatment goal of abstinence.

The earliest of these studies was conducted in 1976 by Orford, Oppenheimer, and Edwards. In this study 100 married, male alcoholics were given either extended outpatient treatment or a single advice session oriented toward abstinence. At the two year follow-up ten men were found to be drinking in a controlled manner and eleven were abstinent. Several significant differences were found between these two groups on pretreatment and treatment variables. One of them was that over half of the abstainers were gamma alcoholics but none of the controlled drinkers were gamma types. This means that the controlled drinkers must have been either alpha or one of the other types

described by Jellinek (1960). Since the arguments and research presented earlier in this review are focused upon the question of whether gamma alcoholics can become controlled drinkers, it is important, then, to look for predictor variables which distinguish between gamma abstainers and gamma controlled drinkers. While comparisons between gamma and alpha alcoholics are perhaps useful, in the context of this review they are peripheral and so this study will not be included in order to maintain theoretical consistency.

Another study, by Miller and Joyce (1979), is similar in that problem drinkers rather than gamma alcoholics were used. Even though significant differences were found between the resultant controlled drinkers and abstainers, due to the theoretical issue mentioned earlier, these results are of limited value to the present discussion and will not be included.

Popham and Schmidt (1976) conducted a study in which a goal of moderate drinking was worked into an outpatient group therapy treatment. Relatively little information is reported on the subjects participating in the research and there is no way to determine whether subjects were gamma or non-gamma type alcoholics. Follow-up information at one year was based entirely on self-report, and no control group was used. For these reasons, the results that controlled drinkers had lower pretreatment consumption of alcohol are

of questionable validity and relevance to the present discussion and so the results of this study will not be included.

Hyman (1976) followed up a group of 20 living, ambulatory subjects 15 years after inpatient treatment for alcoholism. Upon examining the medical records of these former patients he found that the complete abstainers had histories of legal problems, often accompanied by violence, were mostly unmarried, and not regularly employed. Compared to this group, the controlled drinkers showed fewer legal problems, tended to be married, and were regularly employed.

Anderson and Ray (1977) taught self-management skills to an inpatient group of 110 male alcoholics. In their one year follow-up they found that controlled drinkers were older than abstainers (49 vs. 47 years) and pretreatment data on drinking behavior in the year prior to treatment showed that abstainers spent the most days abstinent (54), followed by the controlled drinkers (34), followed finally by the relapsers (11). However, tests of significance apparently were not performed or, if so, they are not reported and so it is difficult to know what these results mean. As a result, they cannot be included in this review.

The research of Vogler et al. (1975) was discussed in the previous section on Controlled Drinking. In this study, the authors also performed a discriminant analysis comparing controlled drinkers with successful abstainers. They found

controlled drinkers had a lower pretreatment alcohol consumption rate.

Vogler, Weissbach, and Compton (1977) employed a discriminant function analysis comparing controlled drinkers with successful abstainers. They found that the former were younger, had a shorter history of drinking problems, had lost fewer days from work in the previous year, and had a lower pretreatment alcohol consumption than abstainers. However, the study cannot be included in this discussion because the comparisons were made using the combined data of outpatients, problem drinkers, and inpatients, thus merging data from gamma alcoholics with other alcoholic types.

Levinson (1977) studied 115 former inpatients five years after discharge. Out of this group he found 11 subjects to be drinking in a controlled manner. In comparing this group to the remainder of the subjects he discovered one significant (statistical data not presented) pretreatment variable, that a greater percentage of controlled drinkers were married at intake. A trend which was not statistically significant was that the controlled drinkers were employed and working full-time. However, for the purposes of this review the comparison was inappropriate as it was made between data from controlled drinkers on the one hand, and the combined data of abstainers and relapsers on the other. The relevant comparison for the purpose of this review is between controlled drinkers and abstainers.

The effect of the relapsers' data is to void the possibility of making the relevant comparison and so even though the results are significant, they are peripheral to this review.

Smart (1978) compared controlled drinkers with abstainers obtained from a population of 1,091 male and female inpatient alcoholics. Specific hypotheses were formulated concerning these two groups. It was hypothesized that in terms of pretreatment variables, controlled drinkers would be younger, drink less, and show better scores on multiple variables, such as scales measuring isolation, physical health, marital stability, etc. The data were analyzed in such a manner as to take into account the multiple comparisons. The results were that prior to treatment controlled drinkers had significantly lower drinking rates and fewer dependence symptoms.

Bromet and Moos (1979) studied 429 alcoholic former inpatients. They divided these subjects into two groups, based upon socioeconomic status, and found 6 months after discharge that 115 subjects were abstaining and 106 were drinking in a controlled manner. Seventy-four variables were used in a search for pretreatment differences between abstainers and controlled drinkers. As Heather and Robertson (1981) point out, with this large number of variables under consideration, at the 5% level of confidence, four differences would be expected to spuriously reach statistical significance by chance alone. They

recommend using the more conservative confidence level of 1% significance. In so doing, two significant pretreatment differences emerge, but only for the low socioeconomic group. Abstainers reported significantly lower numbers of arrests (63 vs. 40%) and lower consumption levels of alcohol (data not reported) prior to intake compared to controlled drinkers.

Polich, Armor, and Braiker (1981) conducted what is considered the most extensive study ever performed on inpatient alcoholics. A group of 2,339 male alcoholics (females and clients referred by the courts for driving while intoxicated were excluded in order to create a more homogenous group) was treated by traditional abstinence methods and followed up at various points in time, up to a maximum of 4 years. At the 4 year mark, 28% of subjects had abstained for the previous 6 months. However, only 7% had abstained for the full 4 years. Eighteen percent of the subjects were classified as controlled drinkers. A regression analysis was performed on variables obtained at intake. The following variables were included: level of dependence symptoms; socioeconomic status; marital status; employment status; age; race; and prior treatment. The analysis was used to estimate the extent to which these variables were associated with higher or lower relapse rates when either abstention or controlled drinking had been adopted by subjects at the 18 month follow-up. The results

reaching statistical significance were that controlled drinkers with an absence of dependence symptoms at intake relapsed less ($p < .05$) than subjects who had abstained 6 months or longer (long-term abstainers). This was also true for several of the other variables, including employment ($p < .05$), age less than 40 ($p < .05$), the presence of low levels of dependence symptoms ($p < .1$), and a marital status of single ($p < .1$). The remaining variables did not yield significant predictions.

A result from the 18 month follow-up (Armor, Polich, & Stambul, 1978) had to do with affiliation with AA. Among a group of subjects who had received a lesser amount of treatment and who had attended AA meetings regularly in the year preceding the follow-up, none were drinking in a controlled manner. For subjects receiving a lesser amount of treatment but who had no AA contact, 31% were controlled drinkers and 16% abstainers. A similar difference was found between subjects in the group receiving a greater amount of treatment. With AA involvement, there were 10% controlled drinkers and 41% abstainers.

Following the pattern of Heather and Robertson (1981), predictor variables with contradictory support (legal problems, marital status, and pretherapy alcohol consumption amounts) will be deleted from further consideration. Further research should be performed to resolve these contradictions. Unlike these authors, however, variables

with only one supporting study have been retained by this author since the difference between having one vs. two supporting studies seems negligible. The four remaining studies show, then, that a controlled drinking outcome is predicted by the following pretreatment variables: a low severity of dependence symptoms (Smart, 1978; Polich et al., 1981); an age of 40 or less (Polich et al., 1981); and regular employment (Hyman, 1976; Polich et al., 1981). An abstinence outcome is predicted by involvement with AA (Armor et al., 1978). It is noted that even with the author's more rigorous selection procedure for inclusion of a study in this section, the resultant predictor variables are identical to those Heather and Robertson (1981) found for mixed populations of gamma alcoholics and non-gamma problem drinkers.

Several issues concerning predictor variables merit further discussion. One issue which should be remembered is that over half of the studies reviewed and all of the studies retained involved an abstinence treatment. The resultant predictor variables thus apply to successful controlled drinkers and abstainers who received an abstinence treatment. It is not clear whether the variables would also apply to successful controlled drinkers and abstainers who had received a controlled drinking treatment. Further, it is possible that other predictor variables would emerge from studies employing a controlled drinking

treatment. Along this line of thinking, Miller and Joyce (1979) speculate that predictor variables generated from studies involving outpatients vs. inpatients and those seeking a controlled drinking vs. abstinence treatment may be dissimilar. And as was true for the studies reviewed in the controlled drinking section, few or no females were used as subjects. Thus, the generalizability of the resultant predictor variables to women is uncertain. Further research is necessary to examine these issues.

Another important consideration in this research is the correlation issue. In the discussion below, the author will speculate that certain of the predictor variables are correlated with each other. If this is the case, then there may be fewer than four distinct variables. Several of these may collapse together. Actually, since the research to date is correlational in nature there is no guarantee that any of the predictor variables is causal of either successful outcome. None, all, or some of these variables may have causal links to these outcomes. Only experimental research in which these variables are used as independent variables in controlled studies will help ferret out which variables are salient causal factors to either a controlled drinking or abstinent outcome. Experimental research is now needed in order to isolate which of these variables (and possibly others) play a crucial, causal role in outcome determination.

Predictor variables: Proposed learning theory explanations. While these variables have emerged as differential predictors of a controlled drinking vs. abstinence outcome for gamma alcoholics, there has been essentially no discussion in the literature of why, on a theoretical basis, these variables would be important. In particular, there is an absence of theorizing how learning theory could specifically be involved. This absence is more conspicuous in light of the research previously reviewed in support of a learning theory alternative to a disease model based upon loss of control. To be consistent, it seems that such theorizing about predictor variables would be a necessary addition to this body of scientific knowledge.

Clearly the problem of alcoholism involves both biological (physical addiction) and psychological (psychological addiction) components (Edwards, 1977). It seems as pointless to try and reduce an understanding of alcoholism to simply biological processes as it does to reduce it simply to psychological processes (cf. Jellinek, 1960, pp. 72; 77). Perhaps it would be more useful to conceptualize a continuum with the label "biological processes" on the left pole and the label "psychological processes" on the right pole. Having done this, it seems reasonable to order the predictor variables along this continuum, with "severity of dependency symptoms" and "age" occupying positions on the left half of the continuum,

"regular employment" near the middle of the continuum, and "AA affiliation" on the right half of the continuum. In this way, each of these variables represents a mixture of biological and psychological components and this ordering reflects the author's thoughts about the relative amounts of each. Intuitively, for example, it seems reasonable to view dependence symptoms as mainly reflecting biological processes and AA affiliation as growing out of a psychological belief or attitudinal system.

If this view of the predictor variables being composed of a mixture of biological and psychological influences is accepted, then we can go on to examine possible specific explanations of the importance of these variables based upon learning theory. Why would a severely dependent alcoholic end up abstinent rather than drinking in a controlled manner? An operant conditioning explanation may be of some value here. Withdrawal symptoms are uncomfortable and severe symptoms can be assumed to be very uncomfortable. Drinking alcohol would act to reduce these withdrawal symptoms, restoring a pleasant state. In this way, drinking is an operant behavior and alcohol consumption is negatively reinforcing, since it causes an unpleasant state to cease. Someone with severe symptoms would need to drink either large amounts of alcohol or else frequently in order to cause the symptoms to abate. The amount of alcohol necessary to control the symptoms may exceed the limit

required for controlled drinking. Thus, in order for the severely dependent alcoholic to be counted as a success in one of the previous studies, he could not have done so by drinking in a controlled fashion but instead must have opted for abstinence. In this way, the principle of negative reinforcement could explain how it is that severely dependent alcoholics are more likely to become abstinent than to control their drinking. The alcoholic with few dependency symptoms, on the other hand, requires less alcohol to diminish the symptoms or perhaps can drink moderately without experiencing any symptoms at all. In this way, for this second case, alcohol imbibing could function as a positive reinforcer since it is not for the purpose of relieving unpleasant symptoms but rather to bring on subjectively perceived pleasant feelings of mild intoxication.

It is difficult to imagine a learning theory explanation of the predictor variable of "age," per se. The extent to which learning theory helps in explaining its importance as a predictor variable is probably a function of some other variable, correlated with age, which is more amenable to a learning theory explanation. It is perhaps not difficult to imagine the younger subjects who went through the treatment programs mentioned in the above studies thinking that it is normal and acceptable to drink. Young people are surrounded by incentives and positive

social reinforcement in many cases to use alcohol. Such a belief and any extant environmental reinforcers could thus bias a younger person toward a controlled drinking option. A young person counted as a success in the above studies would thus more likely be a controlled drinker. At the other end of the spectrum, an older alcoholic, 40 years old or more, could conceivably be thinking about his drinking, "I'm too old to still be doing this. This is not getting me what I want out of life," etc.

A learning formulation for the variable "regular employment" can also be conceived. Someone who is steadily employed at a job has more help in the form of cues and contingencies to remain sober compared to a person who occupies his time in other ways. Positive reinforcement occurs in the form of wages for performing work satisfactorily (viz., soberly). Cognitions such as, "I must keep my job; I must not get fired for drinking," could be present. While it might be thought that these contingencies and cognitions should lead to increased rates of abstinence, it must be remembered that abstinence produced by these factors is meaningful only in comparison to the rate of abstinence produced by the factors present for someone who is not steadily employed. People not regulary employed who were successes in the predictor variable studies may have been more likely to abstain because their environmental contingencies were predisposed toward excessive drinking.

Their social network and the kinds of places available to frequent during the day when others are at work would present more cues and reinforcement for excessive drinking. To try and drink moderately in such an environment would likely be very difficult and so abstinence would be preferred.

"AA affiliation" is the clearest example of how psychological factors in the form of cognitions or mental set could influence an alcoholic toward abstinence. AA ideology exerts a comprehensive influence upon a person's life, including even the spiritual area. When combined with a powerful social support network, AA provides a cognitive structure and much positive reinforcement for staying abstinent. Clearly alcoholics exposed to this ideological perspective would be more likely to abstain than those receiving less or no exposure to it.

Physical Health/Safety Issues

From the above discussion it is seen how a learning theoretical formulation could account for the results of predictor variable studies. However, even if a particular outcome is more probable, based upon the presence of certain predictor variables, it does not necessarily follow that it is also more desirable. Physical health and safety issues are frequently mentioned by a number of authors (Heather & Robertson, 1981; Polich et al., 1981) as important differential variables in selection procedures for a

controlled vs. abstinence drinking outcome. However, the issues have not, in the author's opinion, received the emphasis they deserve in the controlled drinking literature. Following is a discussion of these important issues.

Alcoholism and alcohol abuse are a serious threat to the safety and well-being of citizens and this threat demands effective treatment. Alcohol-related highway crashes kill 25,000 people a year, or half of all the people who die on United States highways. In addition, these accidents seriously injure another 300,000 to 400,000 people and produce colossal expenditures in property damages, policing, court costs, etc. (McCarrol & Haddon, 1962; Bacon, 1973). Further, it has been estimated that between 25-64% of all admissions to medical wards in acute-care facilities are related to alcoholism (Stark & Nichols, 1977). One recent report (Saxe, Dougherty, & Esty, 1983) estimates that up to 15% of the nation's health care costs may be attributed to alcohol abuse and that 85% of those with alcohol problems currently receive no treatment for their condition.

According to deLint and Schmidt (1976) and others (National Institute on Alcohol Abuse and Alcoholism, 1974) there is a firmly established link between the alcoholism of clinically identifiable alcoholics and a higher death rate, compared to the general population. Nicholls, Edwards, and Kyle (1974) found that the death rate for alcoholics is at

least twice that of the general population. Gitlow (1973) reports an overall 12 year reduction in estimated life span for the alcoholic and that 30% of those alcoholics experiencing a severe withdrawal syndrome can expect to expire within 5 years of its occurrence. Alcohol consumption itself is responsible for this increased mortality. The prime causes of these deaths are liver disease (Lelbach, 1974), accidents, and suicide (NIAAA, 1978). These three causes of death show up not only in mortality tables but also in longitudinal studies of alcoholics (Schmidt & deLint, 1972; Pell & D'Alonzo, 1973).

Given the evidence that alcoholism and the consumption of alcohol create tremendous problems both socially and in terms of personal safety and health, it is reasonable to ask if it is known what amounts of alcohol over what period of time are required to produce these results. In the case of traffic accidents it is apparent that difficulty controlling a motor vehicle is a phenomenon associated with consumption of alcohol within a few hours prior to driving and could happen after drinking for the first time. Information from the California Department of Motor Vehicles (1986) indicates that for a person of average weight the consumption of four or five drinks (2-2.5 oz. ethanol equivalent) within a two hour period would give a BAC of .10%, making it "definitely illegal" to drive under California State law. Fewer drinks over the same time period or the same number of drinks over

a longer time period "may be illegal" depending upon other factors such as drinking on an empty stomach or being ill. The chance of having an accident while driving under these above-mentioned conditions is between five and twenty-five times higher compared to non-drinking drivers. Clearly, small amounts of alcohol consumed over relatively short periods of time can under certain circumstances lead to results which are both life-threatening and involve important legal ramifications.

While traffic accidents can occur after a single recent period of drinking, the other physical problems mentioned above require longer periods of sustained drinking in order to be manifest. DeLint and Schmidt (1971), after studying mortality table figures for alcoholics proposed a limit of 150 ml (5 oz.) ethanol equivalent/day as a volume-related definition of alcoholism which entails an extreme risk of organic pathology. Wallgren and Barry (1970) agree that 5 oz. ethanol equivalent/day implies a high risk of liver damage if the drinking is prolonged. An even more conservative figure of 2-5 oz. ethanol equivalent consumed/drinking day is offered by Pequignot, Tuyns, and Berta (1978) and Leiber (1979). Edwards, Chandler, Hensman, and Peto (1972) and Armor et al. (1978) found that when the mean daily consumption of alcohol exceeds 3 oz. ethanol equivalent, the chance of developing withdrawal symptoms such as tremor is higher than when drinking is below this

level. Further, Schmidt and Popham (1975) concluded that when consumption exceeded 12.75 oz. ethanol equivalent/week, there was a significantly raised risk of liver damage. Edwards (1977) reports that this level may be lower for women. This makes sense, given that a woman's lower body weight would lead to higher BAC levels for a given amount of alcohol consumed.

These studies make clear that consuming 5 oz. ethanol equivalent or more/day is definitely unsafe. In light of Schmidt and Popham's (1975), Edwards et al.'s (1972), and Armor et al.'s (1978) findings and the results of Pequignot et al. (1978) and Lieber (1979), 2-5 oz. ethanol equivalent or less/day is probably a more appropriate amount of alcohol which could be consumed without running a significant risk of organic pathology. Even this lower (2 oz.) amount, as noted above, if consumed in a short period of time, would make driving an automobile for a person of average or less weight "definitely illegal" in California and incur an increased risk of having an accident.

Lelbach (1974) in an extensive review of the literature concludes that liver dysfunction can occur after a relatively short time of regular drinking. He reports a 19.2% rate of "severe liver damage" in a group of alcoholics drinking for a period of 1 to 5 years. This rate increases dramatically to 82.4% for those drinking 15 years or longer. Also, for this second group he reports a 51% rate of

cirrhosis, the most severe form of liver damage that can result from drinking. And the liver is not the only organ in the body affected by excessive alcohol consumption. In a study of pancreatic functioning, he reports that consumption of approximately 5 oz. ethanol equivalent/day for 5 years or more led to abnormalities in exocrine pancreatic functioning in 68% of cases. In another study he reports that cardiac myopathy symptoms appeared after a 10-year period of sustained drinking in amounts approximating 8 oz. ethanol equivalent/day.

As can be noted, relatively small amounts of alcohol consumed over a period of from 1 to 15 years can lead to serious organic pathology. In light of this, an important question to ask would be, "What are the operational definitions for amounts of alcohol typically associated with experimental studies in controlled drinking?" Pattison et al. (1977) gave a summary of 74 studies in which controlled drinking was reported as an outcome. In some of these studies this outcome was the specific object of behavioral experimental manipulation but in most it was reported as a spontaneous result of a traditional abstinence treatment program. Of these 74 studies, 28 gave operational definitions of controlled drinking which were quantifiable in terms of ounces of alcohol. Of these 28 studies, 14 reported an operational definition of 1 oz. ethanol equivalent or less/day. The remaining 14 studies were

distributed as follows: five used an operational definition of controlled drinking of 2 oz./day; six used 3 oz./day; one used 4 oz./day; and two used 5 oz./day. Fully half of the studies for which alcohol consumption could be computed meet or exceed the minimum limit proposed by Pequignot et al. (1978) and Lieber (1979) and Schmidt and Popham (1975). Adding the results of seven additional studies (up until 1980) contained in this review for which quantitative operational definitions of controlled drinking could be calculated, these results are revised as follows: fifteen used an operational definition of controlled drinking of 1 oz. ethanol equivalent or less/day; nine used 2 oz./ day; six used 3 oz./day; two used 4 oz./day; two used 5 oz./day; and one used 6 oz./day. While it must be remembered that the studies added since 1977 are a representative but not exhaustive sample, the percentages are revised to just under 60% meeting or exceeding Pequignot et al.'s (1978) and Lieber's (1979), and Schmidt and Popham's (1975) limits.

The reasons why controlled drinking is operationally defined as it is in these studies is unclear. Most likely the values settled upon reflect what is considered realistic in terms of goal-setting for a population which has demonstrated both a wish to continue drinking as well as an inability to regulate their drinking on their own. In the case of the study by Polich et al. (1981) the consumption limits used were arrived at from inspection of population

drinking frequencies from a national survey. Upon this basis, they arrived at a value of 5 oz. ethanol equivalent on drinking days and an overall mean of 3 oz. ethanol equivalent/day. Whatever the reasons, however, it is the author's opinion that a consideration of health and safety needs are of paramount importance and should be foundational in selecting an amount of alcohol consumption appropriate for a controlled drinking regimen. At least one other author has expressed a similar concern (McCrady, 1985). Hopefully, the results of mortality tables and medical studies of alcohol's effects will be incorporated into future research in the area of controlled drinking.

Another important question would be, "Are there differences between abstainers or controlled drinkers in amounts of alcohol consumed?" This is an important issue since subjects in either an abstinence or a controlled drinking training program are likely to struggle with relapse and continued excessive drinking, however temporary it may be. The clearest answer to this question comes from the data of Sobell and Sobell (1978) and the follow-up conducted by Caddy et al. (1978), the results of which were discussed earlier. The operational definition for a controlled drinking day was a maximum consumption of 4.5 oz. ethanol equivalent, and no subject in the experimental group (controlled drinking) reported drinking more than about 4.3 oz. on any day defined as a controlled drinking day. Over

the 3 year follow-up they exceeded the 4.5 oz. limit on just 10% of all days but this declined to about 5% of days in the last year. While the amount of alcohol operationalized as controlled drinking in this study is excessive, as argued above, and while exceeding this amount on 10% of days during the 3 year follow-up is unacceptable for health and safety reasons, these results can only be fairly considered in comparison to the data for the control group (abstinent). In this group, over the 3 years, an average 42.9% of days were spent consuming in excess of the 4.5 oz. limit. The difference in the results between the experimental and control groups is significant at the $p < .01$ level. While it cannot be determined from these results which group actually consumed the greatest total amount of alcohol over time, they do show that the group attempting to be abstinent from alcohol clearly spent more days exceeding the maximum consumption limit, mentioned above, than the group attempting to drink moderately. The conclusion drawn from this study, then, is that in terms of physical health it is safer to be attempting a moderate rather than abstinent drinking goal.

Another study which bears indirectly on this issue is Polich et al. (1981). At the 4 year follow-up three outcome categories resulted: long-term abstainers; short-term abstainers; and drinkers. Only 29% of the short-term abstainers were consuming less than 5 oz. ethanol

equivalent/day. In contrast, 55% of the drinkers fell below this amount. These results also support the conclusion that drinking in moderation is safer than attempting to abstain, at least for short-term abstainers. Further research needs to be done, however, to determine whether this conclusion extends beyond the bounds of these two studies.

Also in this study, mortality for the three identified groups was assessed. At 18 months, relapse was measured by the percentage of alcohol-related deaths. Long-term abstention was superior to short-term abstention (1 vs. 9% deaths) and these differences were statistically significant. Differences between long-term abstainers and controlled drinkers, however, were not significant (1 vs. 3% deaths). It can be seen that the same subpopulation of short-term abstainers that was drinking more also had a higher percentage of deaths than the controlled drinkers. This finding agrees with the findings of Lelbach (1974), discussed earlier, that higher death rates are associated with higher consumption levels of alcohol. More importantly, however, it shows that controlled drinking and long-term abstention are comparable in terms of safety.

A further issue is raised that was noted by Polich et al. (1981). Although the long-term abstainers show the lowest percentage deaths, one cannot reach this stage without first passing through the short-term abstainer category. This poses the question of whether the high

percentage deaths for the short-term abstainers represents all early-stage abstainers in the sample, or whether the short-term abstainers differ in some fundamental way (other than length of abstention) from those who attained long-term abstention. The authors go on to say:

> If the latter case is true, then the low relapse rate for the long-term abstainers would be explained by these other characteristics, rather than by the length of abstention. If the first possibility is true, then the chance of an alcohol-related death for all abstainers would be somewhat higher than the rate... for long-term abstainers. Unfortunately, our data cannot distinguish these two possibilities. Clearly, further investigations of this issue should be carried out. (p. 128)

Further, if the long-term abstainers are simply those short-term abstainers who didn't die over the time period, then the overall percentage of deaths for all abstainers could exceed the percentage for controlled drinkers. This would have important implications for the relative safety of controlled drinking vs. abstinence. The author agrees with Polich et al. (1981) that further research is needed.

Selection of Candidates: Conclusions

A number of conclusions can be reached in this section on the selection of candidates for controlled drinking vs. abstinence. First of all, certain predictor variables have emerged for gamma alcoholics from the literature. Those favoring a controlled drinking treatment are: low severity of dependence symptoms; age 40 or less; and regular employment. The variable favoring an abstinence treatment is involvement with AA. Secondly, while the variables

without question have biological components, they just as clearly have psychological aspects and possible formulations of the variables in terms of learning theory were discussed. Thirdly, certain caveats regarding this research were mentioned, including the uncertainty over whether these and possibly other variables would emerge from future studies employing a controlled drinking treatment, rather than the abstinence-oriented treatment used in all studies from which predictor variables resulted. Also discussed was the correlational nature of the research. This adds uncertainty in terms of the distinctiveness of the variables and their possible causal nature. Further research is needed in which these variables are used as independent variables in studies involving experimental rather than correlational designs. The generalizability of the results to women was also questioned. Further research including more female subjects is needed. Fourthly, it is concluded that an amount of alcohol for controlled drinkers which does not entail the risk of serious organic pathology is no more than 2-5 oz. ethanol equivalent/drinking day, and up to 12.5 oz. ethanol equivalent/week for a male alcoholic. Fifthly, since a significant percentage of studies involving a controlled drinking outcome have used operational definitions of alcohol consumption at the upper end of these limits, or even exceeded them, future researchers should consider revising their operational definitions of controlled

drinking downward. Sixthly, research has been performed suggesting that controlled drinkers, compared to abstainers, may drink to excess less often and have a lower death rate. Further research is needed to validate or disconfirm these findings.

Conclusions

The real issue in the controlled drinking controversy is not whether controlled drinking is a viable alternative to abstinence in the treatment of alcoholism. The viability of this intervention is apparent from the earlier review of controlled drinking research. Rather, it runs deeper than this, to whether alcoholism is better explained by a disease model based upon supposed physiological processes or a model rooted in learning theory. In this Paper, research on loss of control and craving was cited to show that no evidence exists currently for a disease model incorporating these phenomena. Numerous authors have reviewed the literature pertaining to the disease model. Pomerleau (1976) stated that despite a sparseness of evidence the disease model has been treated as though it were established fact. Heather and Robertson found that "no pre-existent biogenetic defect which makes alcoholics in general react differently to alcohol than nonalcoholics has ever been discovered" (1981, p. 249). Milt (1977) examined evidence from studies on nutrition, endocrine functioning, brain damage, differential alcohol metabolism rates for alcoholics vs. nonalcoholics,

and allergic reactions and found that a lack of scientific support exists for the hypothesis of alcoholism as a physical disease. Marlatt (1983; 1985) concludes that there is no evidence of a physiological basis for a disease model of alcoholism.

How can these researchers believe alcoholism is not a physical disease when it is obviously involved in physical deterioration and physical addiction? First of all, the fact that performance of a behavior (drinking) causes physical deterioration (cirrhosis) does not mean necessarily that the behavior itself is a physical disease. Smoking clearly can cause cancer, a disease, but no one (yet!) has called smoking itself a disease. Physical consequences can result from non-physically-based choices. Secondly, and more importantly, physical disease and physical addiction are separate categories of phenomena. Much confusion is caused by blending together or equating these concepts. Heather and Robertson (1981) accuse Keller (1976) of doing this when he cites research pointing to a neurophysiological basis for the Alcohol Dependence Syndrome. Calling alcoholism a disease by equating it with alcohol addiction (dependence) is like calling smoking a disease because it involves, at least to some extent, pharmacological addiction. Along this line, Milt (1977) stated the following:

> Physical dependence (withdrawal) has nothing to do with some supposed 'alteration in cell metabolism,' or with

> some supposed 'tissue demand for the drug.' In fact, nowhere do...pharmacologists themselves hypothesize the existence of these supposed phenomena. The withdrawal symptoms are nothing more than rebound reactions of the central nervous system on release from the depressive influence of the drug. (p. 48)

Thus, it can be seen that physical consequences and physical addiction are separate from the concept of physical disease. Alcoholism involves the first two concepts, but no evidence exists justifying it as a physical disease.

Heather and Robertson (1981) speculate that the traditional demand for permanent abstinence has been based mistakenly on a physical disease model premised upon alcoholics allegedly having an unknown biochemical defect. Instead, as argued above, a more accurate reflection of reality may be that there is no physical disease, only physical addiction. What appears to be an irreversible biochemical defect, unique to alcoholics, and leading to an inexorable loss of control with its affiliated notion of craving is perhaps instead simply the manifestation of physical dependence. But physical dependence symptoms are reversible and can abate over an abstinence period lasting a few days, weeks (Victor & Wolfe, 1973), or months (Heather & Robertson, 1981). Therefore, lifelong abstinence from alcohol is not an absolute requirement in the treatment of alcoholism.

In their Alcohol Dependence Syndrome, Edwards and Gross (1976) postulate the existence of a reinstatement phenomenon. By this it is meant if a severely dependent

alcoholic resumes drinking even after several weeks or months of abstinence, he can become physically dependent again within a relatively short period of time (72 hrs.). It must be stated that this phenomenon is based upon the authors' clinical impression rather than experimental studies with alcoholics. Research is needed to test this hypothesized phenomenon. Even if scientific support for the existence of the reinstatement phenomenon is forthcoming, Heather and Robertson (1981) estimate that 2 weeks to 3 months for a slightly to moderately dependent, and 6 months to 1 year for a severely dependent alcoholic should be sufficient time for the phenomenon to diminish so that a controlled drinking treatment can be attempted if desired and not contraindicated for other reasons.

Overall, the following conclusions can be made. No evidence exists for a disease model of alcoholism based on a conceptualization of loss of control and craving rooted in an irreversible biochemical or other type of physiological defect. No such defect has been discovered to date. On the other hand, behavioral experimental research in the area of loss of control and craving has demonstrated a plasticity and reversibility for the phenomena following the application of interventions based upon learning theory principles, particularly operant and classical conditioning, and cognitive processes. Controlled drinking studies have demonstrated these principles can successfully be applied to

gamma alcoholics.

Confusion has resulted from a tendency to blend together notions of physical disease, the physical consequences of disease, and physical addiction. Alcoholism has debilitating physical consequences and involves physical addiction, but no evidence exists from either behavioral or medical research that it is a physical disease. The hypothesized reinstatement phenomenon, even if shown in the future to be scientifically valid, is thought to be transient and reversible and provides no foundation for an irreversible physical disease model of alcoholism.

Certain predictor variables have emerged from the research. It was found that the variables resulting from a highly select group of studies using gamma alcoholics were identical to those from studies additionally using non-gamma subjects. This argues for wide applicability of the variables. The variables favoring a controlled drinking outcome are: low severity of dependence symptoms; age 40 or less; and regular employment. The variable favoring an abstinence treatment is involvement with AA. It was argued that these variables involve psychological as well as biological aspects and so possible explanations of their salience based upon learning theory were presented.

In the section on physical health/safety it was shown that alcohol consumption operationally defined as controlled drinking was excessive in a significant percentage of

studies pertaining to controlled drinking performed to date. In light of research conducted in the areas of organic pathology, mortality, traffic accidents, and the development of withdrawal symptoms, an amount of alcohol which can safely be consumed should not exceed 12.75 oz. ethanol equivalent/week, up to 2-5 oz. ethanol equivalent/drinking day. Future researchers should revise, if necessary, operational definitions of controlled drinking to fall within these guidelines.

New and continuing research is needed in a number of areas. In the area of application of controlled drinking interventions, it will be necessary to differentiate between various behavioral interventions to see which individual treatment or combination of treatments are more successful. Evidence to date favors a multiple intervention approach.

Also, there is a dearth of research in the application of a behavioral intervention to an abstinence outcome. Since a learning theory model of alcoholism, unlike the disease model, can be applied equally to both controlled drinking and abstinence outcomes, further research should be performed to determine which treatments are conducive to an abstinence outcome. Comparisons could then be made to study differences between behaviorally-based controlled drinking and abstinence treatments.

Female alcoholics have not received sufficient attention in controlled drinking research. In order to

insure the generalization of results of future studies, either greater numbers of women should be involved or else studies using women exclusively should be planned.

In the area of predictor variables, further research is needed to differentiate correlation from causality. Experimental studies using predictor variables as independent variables will hopefully answer this question. Also, since current research on predictor variables is based exclusively upon studies oriented toward abstinence outcomes, it is impossible to know whether the variables emerging from these studies would be the same or different as ones resulting from studies oriented toward a controlled drinking outcome.

The issue of women applies in the area of predictor variables, too. All studies performed to date have either excluded completely or included few females. Future research on predictor variables should include greater numbers of women.

Finally, in the area of physical health/safety issues, more research is necessary to test the tentative finding that controlled drinkers drink to excess less often than abstainers. Some evidence also exists that controlled drinkers have a lower death rate, compared to abstainers. More evidence is needed before firm conclusions can be drawn, however.

Pattison et al. describe the traditional disease model

as a "folk science" (1977, p. 1) and see it as a collection of beliefs, values, and ideologies whose development was more in the service of sociopolitical rather than scientific needs. They believe that a paradigm change is at hand (Kuhn, 1970), from a disease model to a learning theory model. The author, with Heather and Robertson (1981) see this as somewhat premature, since the disease model does not have the scientific backing to justify status as a full-fledged paradigm. Rather, as a "folk science" it better fits Kuhn's (1970) period of prescientific knowledge which precedes the application of the scientific method and paradigm status. Behavioral research (much of which is cited in this Paper) conducted in the area of alcoholism over the nearly quarter of a century since Davies' (1962) study provides a more scientific basis for a paradigm. For this reason Heather and Robertson (1981) propose a new, sociopsychological, learning paradigm for alcoholism. It is a conceptualization of alcohol use and abuse grounded in learning theory, and therefore assumes that alcoholic drinking behavior is essentially modifiable and that no unique differences exist between the drinking behavior of alcoholics vs. nonalcoholics. Given the advancements of psychological learning theory and theoretical research into an understanding of alcoholism and its proven impact on the treatment of alcoholism demonstrated in controlled drinking research, it is time to give full recognition to a learning

theory paradigm of alcoholism which promises to be a fruitful area of further research and source of help to many alcoholics.

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